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QALXANABƏNZƏR VƏZİ DİSFUNKSİYASI İLƏ YANAŞI KEÇƏN 2-Cİ TİP ŞƏKƏRLİ DİABET XƏSTƏLİYİNİN BİOKİMYƏVİ LABORATOR GÖSTƏRİCİLƏRİ

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Xülasə. *Məqalədə 2-ci tip şəkərli diabetin qalxanabənzər vəzi disfunksiyası ilə müşayiət edilməyən və komorbid hipotireozla (HT) və/və ya qeyri-toksik urla (DQTU) keçən hallarında xəstələrin biokimyəvi laborator göstəricilərinin fərqli cəhətlərini öyrənmək məqsədilə aparılmış tədqiqat işi haqqına məlumat verilmişdir. Bu məqsədlə retrospektiv olaraq 596 ŞD2 xəstəsinin xəstəlik tarixləri araşdırılmışdır. Tədqiqatın nəticələri göstərmişdir ki, komorbid HT ilə keçən və ŞD 2 olan xəstələrin qan serumunun lipid profili yüksəksıxlıqlı lipoprotein xolesterolunun aydın ifadə edilən azalması ilə, həmçinin qalıq xolesterolun və triqliseridlərin qalxanabənzər vəzi disfunksiyası ilə müşayiət edilməyən ŞD2 olan xəstələrlə müqayisədə artması ilə xarakterizə edilir. Qalxanabənzər vəzi disfunksiyası ilə müşayiət edilməyən və/və ya diffuz qeyri-toksik uru olan ŞD-li xəstələrdə biokimyəvi profilli laborator göstəricilər arasında aspartatamintransferaza (AST) müstəsna olmaqla, statistic əhəmiyyətli fərq aşkar edilməmişdir. Yalnız qan serumunun AST aktivliyi komorbid HT və DQTU olan xəstələrdə əhəmiyyətli dərəcədə yüksək olmuşdur. Bundan əlavə, ŞD2-nin komorbid HT və ya DQTU ilə müşayiət edildiyi xəstələrdə tireotrop hormonun qan serumundakı konsentrasiyası ilə AST aktivliyi arasında birbaşa korrelyasiya asılılığı müşahidə edilmişdir.*

Açar sözlər: *2-ci tip şəkərli diabet, qalxanabənzər vəzi disfunksiyası, diffuz qeyri-toksik ur*

Ключевые слова*: сахарный диабет 2 типа, дисфункция щитовидной железы, гипотиреоз, диффузный нетоксический зоб*

Keywords*: type 2 diabetes mellitus, thyroid dysfunction, hypothyroidism, diffuse nontoxic goiter*

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BIOCHEMICAL LABORATORY DATA OF TYPE 2 DIABETIC PATIENTS WITH COMORBID THYROID DYSFUNCTION

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The article presents the results of a study conducted to analyze and compare biochemical laboratory data of type 2 diabetic patients without thyroid dysfunction and type 2 diabetic patients with comorbid hypothyroidism (HT) and/or diffuse nontoxic goiter (DNTG). A retrospective analysis of 596 medical records of T2DM patients was carried out.

Serum lipid profile in type 2 diabetes mellitus (T2DM) patients with comorbid HT was characterized by a significant decrease of high-density lipoprotein cholesterol level, as well as an increase of remnant cholesterol and triglyceride levels compared with T2DM patients without thyroid dysfunction, as well as T2DM patients with comorbid DNTG. Biochemical profile data significantly did not differ in T2DM patients without thyroid dysfunction and T2DM patients with comorbid HT and/or DNTG, except aspartate aminotransferase (AST) activity, which was significantly higher in T2DM patients with comorbid HT and DNTG. Moreover, significant direct correlation was observed between serum TSH level and AST activity in T2DM patients with comorbid HT and DNTG.

Diabetes mellitus (DM) is a multifactorial metabolic disorder, which became a global health problem. The number of people with diabetes in Ukraine is growing every year and currently more than 1.3 million DM patients are enrolled in an official registry, however, the total number of diabetics in the country may reach 3.5 million people [1]. Complications of type 2 diabetes mellitus (T2DM) are detected in more than 80% of patients, of which 50% have two or more complications. Excessive body weight, obesity, dyslipidemia and hypertension have been found to contribute to further risk of T2DM and its complications [2]. In recent years, researchers have paid more attention to the comorbid course of T2DM with thyroid dysfunction [3]. Among patients with T2DM, thyroid dysfunction is more common than in the general population. The prevalence of thyroid dysfunction among patients with T2DM has been reported to range from 2.2 to 17.0% [4]. Hypothyroidism (HT) and diffuse non-toxic goiter (DNTG) are common thyroid disorders. In regions with sufficient iodine intake, the prevalence of primary HT ranges from 1.0 to 2.0% [5]. On the other hand, in patients with T2DM, according to various authors, the prevalence of HT ranges from 5.7 to 37.1% [6, 7]. The prevalence of DNTG increases with increasing iodine deficiency and becomes endemic in populations where iodine intake is insufficient [8]. Thus, in the world population, the prevalence is 15.8%, ranging from 4.7% in America to 28.3% in Africa [8-10]. Recently, routine laboratory investigations, including serum biochemical profile, have attracted attention in their potential use for adverse outcomes risk stratification in diabetic patients, especially in comorbid course of T2DM.

The aim of our study was to analyse and compare serum biochemical profile of type 2 diabetic patients without thyroid dysfunction and type 2 diabetic patients with comorbid HT and/or DNTG.

Materials and methods. We made a retrospective analysis of 596 medical records of the type 2 diabetic patients who were hospitalized to the Endocrinology department of the municipal non-profit enterprise "Ternopil University Hospital" of Ternopil Regional Council in 2019. Patients were divided into 4 groups: group 1 (501 patients without comorbid thyroid dysfunction), group 2 (37 patients with comorbid HT), group 3 (40 patients with comorbid DNTG) and group 4 (18 patients with comorbid HT and DNTG).

The diagnosis of T2DM was confirmed according to the recommendations of the American Diabetes Association [11]. HT was diagnosed according to the criteria of the European Thyroid Association [12]. If T4 values were within normal limits, subclinical hypothyroidism (SCH) was diagnosed. The diagnosis of DNTG was confirmed according to the WHO guidelines [13].

Patients with a history of other thyroid diseases (than HT and DNTG), patients which were prescribed thyroid hormone-related drugs, patients with pregnancy or lactation, as well as with cancer, infectious diseases, neurological or mental diseases (depression, anxiety and schizophrenia) were excluded from the study.

Thyroid sonography was performed for all participants that included transverse and longitudinal location.

Serum biochemical profile (total protein, total amylase, urea, creatinine, total bilirubin, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) in blood serum was determined using a standard kits with a COBAS INTEGRA® Diagnostics automatic biochemical analyser in the Biochemical Laboratory of Ternopil University Hospital, Ternopil, Ukraine. Serum lipid profile (total cholesterol (TC), triacylglycerols (TG) and high-density lipoprotein cholesterol (HDL-C)) were determined with commercially available kits on a Cobas 6000 analyzer (Roche Hitachi, Germany) in the Biochemical Laboratory of Ternopil University Hospital, Ternopil, Ukraine. Friedewald's formula was used to calculate low-density lipoprotein cholesterol (LDL-C) levels: LDL-C (mmol/L) = TC – HDL-C – (0.45 × TG). Non-HDL-cholesterol was calculated using the formula: non-HDL-C = TC – HDL-C. Remnant cholesterol (RC) was calculated using the formula: RC (mmol/L) = TC – (HDL-C + LDL-C).

Plasma insulin level was determined by the help of enzyme-linked immunosorbent analyzer "Thermo Scientific Multiskan FC" using DRG set (Germany). HOMA-IR (Homeostasis Model Assessment for Insulin Resistance) index was used to determine IR. It was calculated using the formula: HOMA-IR= (fasting plasma glucose, mmol/l × fasting plasma insulin, μIU/ml)/22.5 [14].

Body mass index (BMI) was calculated using the formula: body weight (kg) / height (m^2).

Study results were analysed using STATISTICA 7.0. The Kolmogorov–Smirnov test was used to compare probability distributions. Quantitative values, due to their non-parametric distribution, are compared using the Mann–Whitney test and were presented as median (Me) and interquartile range. The results were considered statistically significant at the probability level (p-value) <0.05. The association between the studied indices was established based on the results of the correlation analysis using Spearman's rank correlation coefficient.

Results and discussion. Analysis of the data of type 2 diabetic patients included in the

Table 1. *The indices of carbohydrate metabolism in T2DM without comorbid thyroid dysfunction and type 2 diabetic patients with comorbid HT and/or DNTG, Median (Min; Max)*

Group	Glucose, mmol/l	HbA1c, %	Insulin, µIU/ml	HOMA-IR
T2DM	9.4 (7.5;12.1)	8.1 (6,99;9.3)	13.85 (9.87;17.34)	5.59 (4.58;6.87)
p_1	0.9751	0.00528*	0.3124	0.1383
T2DM + HT	9.92 (8.04;12.4)	9 (7.47; 10)	14.32 (10.25;17.15)	6.09 (4.52;7.71)
p_2	0.6216	0.1537	0.5009	0.8085
T2DM + DNTG	8.49 (7.84;10.18)	8.7 (7.6;10.09)	15.02 (10.75;18.71)	5.85 (4.56;6.72)
p_3	0.4441	0.1603	0.8863	0.1868
T2DM + HT + DNTG	9.44 (7.92;10.71)	9.75 (8.05;11.09)	16.28 (11.67;17.31)	6.44 (5.94;7.06)

Note: p_1 - T2DM vs T2DM + HT + DNTG, p_2 - T2DM + HT vs T2DM + HT + DNTG, p_3 - T2DM + DNTG vs T2DM + HT + DNTG; * - statistically significant difference.

study showed that the average age of patients in the group T2DM was 56 (50; 62) years, 56 (52; 61) years in the group T2DM + HT, 58 (55; 64.25) years in the group T2DM + DNTG and 58 (57; 68) years in the group T2DM + HT + DNTG. BMI in the group T2DM + HT + DNTG was significantly higher by 15.88% compared with the group T2DM without thyroid pathology, by 11.77% compared with the group T2DM + HT and by 12.41% compared with the group T2DM + DNTG.

The evaluation of carbohydrate metabolism (table 1) revealed significantly higher levels of HbA1c by 20.37% in the group T2DM + HT + DNTG compared with only T2DM group. Comparing data of insulin, fasting glucose and HOMA-IR index, no significant differences were found between the study groups.

Evaluating the data of the serum lipid profile (table 2), we found significantly higher levels of RC (by 64.00%) and lower levels of HDL-C (by 23.63%) in the group T2DM + HT + DNTG compared with T2DM only group. There was also a 49.73% increase of TG level in patients with comorbid T2DM, HT and DNTG compared with T2DM only patients. Significantly higher levels of RC (p=0.01436) and TG (p=0.0144) were also found in the group T2DM + HT + DNTG compared to T2DM + DNTG group.

Table 2. *The indices of lipid metabolism in type 2 diabetic patients without comorbid thyroid dysfunction and T2DM with comorbid HT and/or DNTG, Median (Min; Max)*

Group	TC, mmol/L	HDL-C, mmol/L	LDL-C, mmol/L	RC, mmol/L	non-HDL-C, mmol/L	TG, mmol/L
T2DM	4.99 (4.28;5.88)	1.1 (0.95;1.24)	3.05 (2.4;3.9)	0.75 (0.44;1.18)	3.92 (3.21;4.68)	1.83 (1.09;2,8)
p_1	0.8334	0.0003678*	0.4941	0.0003299*	0.2785	0.008401*
T2DM + HT	5.28 (4.8;5.96)	0.98 (0.82;1.17)	3.34 (2.64;3.91)	1.06 (0.72;1.29)	4.5 (3.74;4.88)	2.53 (1.9;2.92)
p_2	0.1694	0.1866	0.09682	0.09306	0.346	0.1641
T2DM + DNTG	5.25 (4.72;5.98)	1.04 (0.82;1.32)	3.2 (2.75;4.07)	0.96 (0.64;1.2)	4.06 (3.58;4.83)	2.14 (1.41;2.66)
p_3	0.4145	0.07049	0.1504	0.01436*	0.7685	0.0144*
T2DM + HT+DNTG	4.96 (4.8;5.6)	0.84 (0.78;1.02)	2.93 (2.6;3.34)	1.23 (1.14;1.31)	3.96 (3.74;4.58)	2.74 (2.53;2.92)

Note: p_1 - T2DM vs T2DM + HT + DNTG, p_2 - T2DM + HT vs T2DM + HT + DNTG, p_3 - T2DM + DNTG vs T2DM + HT + DNTG; * - statistically significant difference

During the study of serum biochemical profile we found significant changes only in one parameter – AST activity in the group T2DM + HT + DNTG – 25.2 (22; 30.6) U/l compared to T2DM + DNTG –18.2 (13.25; 24.68) U/l (p=0.02819) and compared to T2DM only – 18.1 (14.2; 25.8) U/l (p=0.01379). No statistically significant difference was found between other studied biochemical parameters in type 2 diabetic patients without comorbid thyroid dysfunction and type 2 diabetic patients with comorbid HT and/or DNTG.

When evaluating the level of thyroid hormones, significantly lower TSH levels were obtained in the group of type 2 diabetic patients without thyroid dysfunction compared to the group of T2DM + HT + DNTG ($p<0.001$) and significantly higher by 64.9% T4 levels ($p<0.001$), respectively. There are also higher levels of TSH ($p<0.001$) and lower levels of T4 ($p<0.001$) in the group T2DM + HT + DNTG compared with T2DM + DNTG group.

An increase in the size of the thyroid gland in the group T2DM + HT + DNTG compared with T2DM only and T2DM + HT groups was found by the help of ultrasound investigation. Thus, the total thyroid volume was by 47.41% and by 51.32% significantly higher in the group T2DM + HT + DNTG compared with the groups T2DM only and T2DM + HT, respectively.

We analyzed correlations between serum TSH and T4 levels and HbA1c, TG levels and AST activity in all study groups. Significant direct correlations were observed only between serum TSH level and HbA1c ($r=0.42$, $p=0.031$), TG levels ($r=0.34$, $p=0.048$) and AST activity ($r=0.38$, $p=0.042$) in type 2 diabetic patients with comorbid HT and DNTG.

Thyroid dysfunction and T2DM often tend to coexist in patients [15-17]. There are data available that the prevalence of HT in T2DM patients ranges from 6.0% to 20.0% across different ethnic groups [3, 6]. On the contrary, M. Smithson reported lower prevalence rates for thyroid dysfunction in type 2 diabetic patients [18]. These inconsistencies could be explained by differences in age, sex, and iodine intake in the populations surveyed [19]. Undiagnosed thyroid dysfunction may affect the metabolic control and enhance cardiovascular, and other chronic complication risks in diabetic patients [20], therefore, it is very important to determine risk factors for thyroid dysfunction development among T2DM patients.

The evaluation of glucose metabolism indices established significantly higher levels of HbA1c in the group T2DM + HT + DNTG compared with T2DM only group. Barmpari et al. reported higher levels of HbA1c in patients with T2DM and HT compared with diabetic patients without HT [7]. According to Kim et al. HT falsely raises HbA1c due to decreased erythropoiesis [21]. Thyroid hormone replacement is associated with a decrease in HbA1c level, which is influenced by increased erythropoiesis rather than by changes in glucose level. Moreover, we found significant direct correlation between serum TSH level and HbA1c in type 2 diabetic patients with comorbid HT and DNTG. T. Karar et al. showed a weak positive correlation ($r=0.212$, $P=0.034$) between HbA1c and TSH levels in type 2 diabetic patients [22].

Our result of a dyslipidaemia in patients with T2DM and comorbid thyroid dysfunction is consistent with the results by Du et al. who identified low levels of HDL-C in T2DM patients as a risk factor for thyroid dysfunction [23]. Elgazar et al. [24] also reported in their study a significant increase of TG levels in T2DM patients with comorbid thyroid dysfunction compared with T2DM only patients.

X. Wanjia et al. reported that TSH levels were correlated in a positive linear manner with the TC and TG levels in Chinese population with newly diagnosed asymptomatic coronary heart disease [25]. L. Tian et al. indicated that TSH might up-regulate hepatic 3-hydroxy-3-methyl-glutaryl coenzyme A reductase expression, which suggested a potential direct role of TSH in the cholesterol biosynthesis in the liver [26]. X. Wanjia et al. found that the TSH level was significantly higher in the hypercholesterolemic and hypertriglyceridemic subjects vs patients with normal levels of TC and TG [25]. Similar results have been obtained by

Lai et al. [27], who demonstrated that the TSH level in the hypertriglyceridemia group was much higher than in the normal control group. In case of lipid profiles, the concentrations of TC, TG and non-HDL-C were significantly higher in patients whose TSH level were in the upper limits than those whose TSH levels were in the lower limits of the normal range. This phenomenon was supported by the HUNT study [28], which suggested that within the clinically normal TSH range, the increasing level of TSH was associated with less favorable lipid concentrations.

Evaluating the data of serum biochemical profile, we found significant changes only in one parameter – AST activity. A.M. Kucharska et al. analysed biochemical laboratory data of patients with severe HT due to autoimmune thyroiditis [29]. They observed a preponderance of AST elevation: it was detected in 82% of patients and ALT increased in 65%. The value of TSH correlated positively only with AST activity. Most researchers suggest that AST elevation in HT results from associated myopathy, not only the liver injury. On the other hand, other factors could also be involved in pathophysiology of the injury, including oxidative stress and decreased ceruloplasmin level, which is reported in hypothyroid patients [30].

Conclusions

1. Type 2 diabetic patients with comorbid HT and/or DNTG had significantly higher BMI and increased level of HbA1c compared with diabetic patients without thyroid dysfunction.
2. Serum lipid profile in T2DM patients with comorbid HT was characterized by a significant decrease of HDL-C level, as well as an increased of RC and TG levels compared with T2DM patients without thyroid dysfunction, as well as T2DM patients with comorbid DNTG.
3. Biochemical profile data significantly did not differ in T2DM patients without thyroid dysfunction and T2DM patients with comorbid HT and/or DNTG, except AST activity, which was significantly higher in T2DM patients with comorbid HT and DNTG. Moreover, significant direct correlation was observed between serum TSH level and AST activity in T2DM patients with comorbid HT and DNTG.

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БИОХИМИЧЕСКИЕ ЛАБОРАТОРНЫЕ ДАННЫЕ БОЛЬНЫХ САХАРНЫМ ДИАБЕТОМ 2 ТИПА С КОМОРБИДНОЙ ДИСФУНКЦИЕЙ ЩИТОВИДНОЙ ЖЕЛЕЗЫ

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Резюме. В статье представлены результаты исследования, проведенного с целью анализа и сравнения биохимических лабораторных данных больных СД 2 без дисфункции щитовидной железы и больных сахарным диабетом 2 типа (СД2) с коморбидным гипотиреозом (ГТ) и/или диффузным нетоксическим зобом (ДНТЗ). Был проведен ретроспективный анализ 596 историй болезни больных СД2.

Результаты исследования показали, что липидный профиль сыворотки крови больных СД2 с коморбидным ГТ характеризовался достоверным снижением уровня холестерола липопротеинов высокой плотности, а также повышением уровня остаточного холестерола и триглицеридов по сравнению с больными СД2 без дисфункции щитовидной железы, а также с больными СД2 с коморбидным ДНТЗ. Данные биохимического профиля у больных СД2 без дисфункции щитовидной железы и у больных СД2 с коморбидным ГТ и/или ДНТЗ достоверно не различались, за исключением активности аспартатаминотрансферазы (АСТ), которая была достоверно выше у больных СД2 с коморбидным ГТ и ДНТЗ. Кроме того, наблюдалась достоверная прямая корреляция между уровнем ТТГ в сыворотке крови и активностью АСТ у больных СД2 с коморбидным ГТ и ДНТЗ.

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